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are a weak form of low-carb strategy

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problem. The glyccmic load (GL) is defined as the GI multiplied

by the grams of carbohydrate in a sample of a particular food.

Obviously, GL is still an intensive variable. You still have to

know how much is consumed. More important, are you really

sure that GL will really be different from total carbohydrate,

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There is also the overall character of using GL A slice of

white bread has a high GI. The GI will go down if you smear a

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if you add two tablespoons of butter. If you could somehow butter infinitely, until for all intents and purposes you have pure butter, you would have a GI = 0, which is probably not helpful for those who want to use the GI as a guide to eating.

One final ambiguity: GI measures blood glucose. Fructose, a sugar of great current interest (because it is 50 percent of sucrose and slighdy more than 50 percent of high-fructose corn syrup), is partially converted to glucose in two hours, which is why the GI of fructose is 20 and not zero. In fact, more is converted after that time, severely compromising any assertion about the diff er¬ences in effect of the two sugars. Sucrose has a GI of 70, wliich is roughly the average of glucose and fructose. Thus, ice cream has a lower GI than potatoes. Yet now we cant recommend ice

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The original questionnaire given to medical students asked one question about the glycemic index. Intellectually, the glycemic index was an important idea. It followed the same principle as low-carbohydrate diets, and was seemingly of practical value. The intention of the glycemic index was to address the experi¬mental effect of carbohydrate on blood glucose. The glycemic index addresses the old idea, pretty much a dogma when I was in school, that simple sugars would cause a rapid rise in blood glucose, but complex carbohydrate—which at that time still meant polysaccharides (starch)—would not. The idea was ques¬tioned at some point, however, and it turns out that when you actually measure the effect of foods on blood glucose, it's not easily predictable—that is, it must be determined experimen¬tally. Glycemic index (GI) is precisely defined as the area under the blood glucose time curve during the first two hours after consumption of 50 grams of carbohydrate-containing food. In other words, it is the total amount of blood glucose for a fixed time period after ingestion.

Whatever its promise, low-GI diets have evolved to be a politically correct form of carbohydrate restriction, and it is

questionable if they have any value at all. Eric Westman, who has experience with both kinds of diets, put it well: “If low-GI is good, why not no-GI?”

GI is mainly influenced by the absolute concentration of glucose in the food, the extent to which glucose appears in the blood (not necessarily from the food itself), and the quantity of other nutrients such as fat or fiber in the carbohydrate containing food that might slow the digestion or absorption of carbohydrate. The individual personal variation makes it

Whaddaya Know?

doubtful that GI diets are useful at all. In comparison to simply reducing carbohydrate, low-GI strategies are complicated and require looking up and calculating values, a feature that might be appealing to some, but is probably annoying to most.

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cream because of the high fructose. Lower GI or lower fructose? How can you do both without saying “low-carbohydrate” out loud? Tliis tangled web is woven out of the failure to face scien¬tific facts. This aspect of the nutritional crisis is probably best addressed by ignoring glvcemic index altogether.

The work of Volek and Forsythe provides a good reason to focus on the carbohydrate content of your diet. What about the type of carbohydrate, though? In other words, is glycemic index important? Is fructose as bad as they say? Consistent with the small perturbation caused by fructose compared to glucose, as shown in the previous chapter, we have a good general principle: No change in the type of macro nutrient—carbohydrate or fat—will ever have the same kind

of effect as replacing carbohydrate across the board with fat.

Hunger

What It Is, What to Do About It

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glycemic index diet. 3 As I explained in chapter 2, the glycemic index

is a measure of the actual effect of dietary glucose on blood glucose.

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rationale as a low-carbohydrate diet: that glycemic and insulin fluctua¬

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Diabetes

After the completion of the twenty-four-week studyjenkins concluded:

“In patients with type 2 diabetes, 6-month treatment with a low-glycemic

index diet resulted in moderately lower HbAlc levels compared with a

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Jenkins’s study came out, Eric Westman’s group published a study that

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ies were comparable in duration and number of subjects, and a direct

comparison (figure 10.1) shows that the true low-carbohydrate has much

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Glycemic Index

Politically Correct Low-Carbohydrate

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question about the glycemic index. Intellectually, the glycemic index was an important idea. It followed the same principle as low-carbohydrate diets, and was seemingly of practical value. The intention of the glycemic index was to address the experi¬ mental effect of carbohydrate on blood glucose. The glycemic index addresses the old idea, pretty much a dogma when I was in school, that simple sugars would cause a rapid rise in blood glucose, but complex carbohydrate—which at that time still meant polysaccharides (starch)—would not. The idea was ques¬ tioned at some point, however, and it turns out that when you actually measure the effect of foods on blood glucose, it's not easily predictable—that is, it must be determined experimen¬ tally. Glycemic index (GI) is precisely defined as the area under the blood glucose time curve during the first two hours after consumption of 50 grams of carbohydrate-containing food. In other words, it is the total amount of blood glucose for a fixed time period after ingestion.

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Health, and National Institutes of Health (NIH)—has been on an antifat crusade for fifty years. No experiment will make them change their point of view—or more precisely, clearly describe their point of view, which they can apparently mold and reform to fit any challenge. Low-fat products are still everywhere and low-fat is still recommended in one way or another.

The diet-heart hypothesis holds that lipid in your diet, particularly satu¬

rated fat, will raise one or another lipid fraction in your blood, which will

in turn cause you to have a heart attack. TheyVe put it to the test—big,

expensive clinical trials with tens of thousands of subjects, hundreds of

millions of dollars—and it consistently fails. To figure out whats going on,

and to avoid the path to poor health, you have to understand the details.

Some of the details involve more chemistry. Many terms in the popular

media are used incorrectly and contribute to the current crisis. A little

precision will help you understand the problem.

First, you need to know that the term lipid refers to a diverse collection

of chemical compounds, all of which are sparingly or not at all soluble in

water. The group includes fatty acids, fats and oils, cholesterol, and deriva¬

tives of these compounds. Directly applicable here are the fats and oils and

their constituent components, the fatty acids and glycerol. WeTl look at fat

structure and the meaning of saturated and unsaturated fat.

The crux of the crisis is the packaging of lipids into complex units, the

lipoproteins (LDL, HDL, and others) that transport cholesterol and fat.

These particles are targeted as surrogate markers for cardiovascular risk.

As surrogates, they are of limited value. The extent to which they truly

indicate risk is controversial. Much of the revolution in nutrition revolves

around facing the fact that a surrogate is not the same as actually getting

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sick. It is still important to be familiar with them, however. They will be

part of your lipid workup when you get a clinical lab test.

The big payoff will be wrapping your head around how fat interacts with

carbohydrate, and looking ahead, we will try to understand how carbohy

drate can be converted to fat, but, to

a large extent, fat cannot be converted

to glucose. We will want to grasp how it is that we cannot use our fat stores

to keep glucose at normal levels and how it is that the amount of dietary

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greater benefits—hence the quotation from Dr. Westman at the head of

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Figure 10.1 by itself constitutes the best evidence for a low-carbohydrate

diet as the "default diet," the one to try first, for diabetes and metabolic